

# The Association between Caries and Childhood Lead Exposure

James R. Campbell,<sup>1</sup> Mark E. Moss,<sup>2</sup> and Richard F. Raubertas<sup>3</sup>

<sup>1</sup>Department of Pediatrics, <sup>2</sup>Department of Community and Preventive Medicine, and <sup>3</sup>Department of Biostatistics, University of Rochester School of Medicine and Dentistry, Rochester, New York, USA

Epidemiologic studies suggest an association between lead exposure and caries. Our objective was to establish whether children with a higher lead exposure as toddlers had more caries at school age than children with a lower lead exposure. We used a retrospective cohort design. A sample of children who attended second and fifth grades in the Rochester, New York, public schools during the 1995–1996 and 1996–1997 school years were examined for caries through a dental screening program. For each child we assessed the number of decayed, missing, or filled surfaces on permanent teeth (DMFS), and the number of decayed or filled surfaces on deciduous teeth (dfs); the number of surfaces at risk (SAR) was also recorded. Lead exposure was defined as the mean of all blood lead levels collected between 18 and 37 months of age by fingerstick [provided the blood lead level was  $\leq 0.48 \mu\text{mol/L}$  ( $\leq 10 \mu\text{g/dL}$ )] or venipuncture. A total of 248 children (197 second graders and 51 fifth graders) were examined for caries and had a record of blood lead levels to define lead exposure. The mean dfs was 3.4 (range 0–29); the mean DMFS was 0.5 (range 0–8). Logistic regression was used to examine the association between the proportion of children with DMFS  $\geq 1$ , and the proportion with dfs  $\geq 1$ , and lead exposure [ $< 0.48 \mu\text{mol/L}$  vs.  $\geq 0.48 \mu\text{mol/L}$  ( $< 10 \mu\text{g/dL}$  vs.  $\geq 10 \mu\text{g/dL}$ )] while controlling for SAR, age at examination, and grade in school. For DMFS, the adjusted odds ratio was 0.95 [95% confidence interval (CI), 0.43–2.09;  $p = 0.89$ ]; for dfs, the odds ratio was 1.77 (95% CI, 0.97–3.24;  $p = 0.07$ ). This study did not demonstrate that lead exposure  $> 10 \mu\text{g/dL}$  as a toddler was a strong predictor of caries among school-age children. However, the results should be interpreted cautiously because of limitations in the assessment of lead exposure and limited statistical power. **Key words:** blood lead, children, dental caries, dentistry, lead poisoning, teeth. *Environ Health Perspect* 108:1099–1102 (2000). [Online 27 October 2000]

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Despite declines in recent years, the prevalence of elevated blood lead levels remains a concern in the United States. The second part of the Third National Health and Nutrition Examination Survey (NHANES III), conducted from 1991 to 1994, estimated that 4.4% of children 1–5 years of age have blood lead levels  $\geq 0.48 \mu\text{mol/L}$  ( $\geq 10 \mu\text{g/dL}$ ) (1). Thus, approximately 890,000 children in the United States (2) have blood lead levels exceeding the threshold defined by the Centers for Disease Control and Prevention (CDC) (3). Many of these children are urban, minority children (1,3,4). This same population of children also has the highest rates of dental caries in the United States (5,6). This disproportional burden of caries and lead exposure in urban, minority populations suggests a potential association.

Results of animal-model studies report an association between lead exposure and caries (7,8). In a recent study, pregnant rats were randomized to receive either lead-contaminated water or lead-free water; the exposure was continued until the rat pups were weaned (9). The mean smooth surface and sulcal surface caries scores were higher among the lead-exposed rat pups than the nonexposed rat pups.

Human epidemiologic studies also report an association between lead exposure and

caries. Studies report a positive association between lead level in teeth and caries (10,11). Gil et al. (12) reported that a high tooth lead level is significantly associated with levels of plaque and Lactobacilli (odds ratio 2.79 and 2.52, respectively), both known risk factors for caries. However, this study was cross-sectional, and thus could not establish the sequence of exposure and disease.

We conducted a study to examine the temporal association between lead exposure and caries. In contrast to the study of Gil et al. (12), our study used blood lead measurements made when the subjects were toddlers, a critical time period in permanent-tooth development. Our objective was to determine whether children with higher lead exposure as toddlers had more caries at school age than children with lower lead exposure.

## Materials and Methods

### Identification of Subjects

We used a retrospective cohort design. Children who attend second and fifth grades in the public schools in the city of Rochester, New York, are examined for caries through a program conducted by the Eastman Dental Center. The caries measures of all children examined are maintained on a computerized database. All subjects in this

study were identified from the 1995–1996 and 1996–1997 academic school years of this database.

We linked the children in the caries database to a database of blood lead levels maintained by the local county health department. The blood lead level database is a record of all blood lead levels obtained on children who resided in Monroe County, which includes Rochester, New York, since 1986. We excluded entries for children born before March 1984 and after September 1990 to restrict the database to children who could have been second or fifth graders during the 1995–1996 and 1996–1997 academic school years. We also excluded blood lead levels obtained on children less than 18 months of age or greater than 37 months of age, and all fingerstick blood lead levels  $\geq 0.48 \mu\text{mol/L}$  ( $\geq 10 \mu\text{g/dL}$ ). The names (first and last), health department identification number, and date of birth of the remaining children were compiled onto a roster; blood lead levels were not included on this roster. One investigator (J.C.) searched the roster for each child examined for caries by the caries screening program. Individuals on the roster and on the caries database were compared by first name, last name, month of birth, day of birth, and year of birth; at least four of these identifiers had to match to allow enrollment into the study. Children who were enrolled constituted the primary sample of the study.

The Institutional Review Board of the Eastman Dental Center and Strong Memorial Hospital approved this study.

### Measures

**Lead exposure.** For each child, lead exposure was defined as the mean of all blood lead levels which met the following criteria: *a*) collected between ages 18 and 37 months and *b*) collected via venipuncture phlebotomy or

Address correspondence to J. R. Campbell, Rochester General Hospital, Department of Pediatrics, MOB Suite 300, 1425 Portland Avenue, Rochester, NY 14621-3095 USA. Telephone: (716) 922-4028. Fax: (716) 922-3929. E-mail: James.Campbell@viahealth.org

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via fingerstick phlebotomy, provided the blood lead level was  $\leq 0.48 \mu\text{mol/L}$  ( $\leq 10 \mu\text{g/dL}$ ). The first criterion defines the age period during which blood lead levels tend to peak in children (13); selecting blood lead levels from this age period thus maximizes the variance of the lead exposure measure. The second criterion was necessary because blood collected by fingerstick phlebotomy may be contaminated by lead-containing dirt on the skin (3).

Laboratories in Monroe County are accredited to assay blood for lead by the New York State Department of Health and are required to participate in a proficiency-testing program (14). They are required to report demographic information, phlebotomy method (fingerstick or venous), and the blood lead level of all children under 6 years of age to the local county health department.

Blood lead levels were measured by graphite furnace atomic absorption spectrometry (15). Blood samples were assayed twice and the mean was reported.

Lead exposure is ideally based on multiple blood lead levels obtained over a period of time (16), or on bone or tooth lead level measured by X-ray fluorescence (17). Nevertheless, studies demonstrate that a single blood lead level at age 24 months is correlated with a cumulative lead exposure measure at early school age, the age period during which the caries assessment was conducted in our study. Rabinowitz et al. (18) reported a moderate correlation ( $r = 0.48$ ) between blood lead level at age 24 months and dentine lead level in deciduous teeth at age 7 years. Dietrich reported a high correlation ( $r = 0.86$ ) between the blood lead level at age 24 months and the mean lifetime blood lead level (defined as the mean of 20 quarterly blood lead levels from age 3 months to 5 years) (19).

**Dental caries.** For each child we defined caries as the number of decayed or filled surfaces on deciduous teeth (dfs), and the number of decayed, missing, or filled surfaces on permanent teeth (DMFS). The data for these measures were obtained from an examination conducted through a caries screening program, described above. The caries screening examination was done by licensed hygienists who were unaware of the child's lead exposure status. The visual-tactile criteria of Radike was used for determining caries (20); no radiographs were used. Caries were scored with the aid of a fiber-optic light source, a plane mirror, and a no. 23 piano wire explorer. At the time of the caries assessment, the hygienist also obtained a plaque score and recorded whether each tooth surface was sealed.

**Measurement of covariates.** After identifying the primary sample, we obtained

measures of covariates via a telephone interview. The interviewers were blinded to the child's caries and lead exposure status. The interviewers called homes based on telephone numbers obtained from three urban pediatric clinics in Rochester and from the Rochester City School District. The interviewers called on multiple occasions during the day, evening, and weekends. If no contact was made after at least five attempts, the interviewers mailed a letter to the parents asking them to call.

The questionnaire used in the telephone interview had five sections that asked about demographics, fluoride exposure, diet, oral hygiene, and medical history (Table 1). Regarding demographics, we collected the head of household's occupation and highest grade attained in school in order to calculate socioeconomic status as defined by the Hollingshead scale (21). The child's date of birth, sex, and ethnicity were obtained from the caries database.

In addition to collecting data on tooth brushing and flossing, oral hygiene was also assessed by the hygienist as part of the dental examination. Hygiene was ranked as "good" if there was no plaque and "poor" if the degree of plaque buildup required a referral for cleaning; all other degrees of hygiene were ranked as "fair." The hygienist also assessed each tooth surface to determine whether it was sealed and therefore not at-risk for caries. We defined surfaces at risk as the number of tooth surfaces present minus the number of tooth surfaces that were sealed.

### Analysis

We analyzed caries experience separately for permanent teeth (DMFS) and deciduous teeth (dfs). For the analysis of DMFS, children with no permanent teeth were excluded, and for analysis of dfs, children with no deciduous teeth were excluded.

The distribution of caries was highly skewed, with at least half of the subjects having DMFS or dfs = 0 (see "Results"). Therefore, logistic regression was used for analysis, with the presence or absence of caries as the response. Explanatory variables included the number of surfaces at risk, age at exam, grade in school (2 or 5), and lead exposure. Lead exposure was dichotomized at a mean blood lead level of  $0.48 \mu\text{mol/L}$  ( $10 \mu\text{g/dL}$ ), using the cutoff defined by the Centers for Disease Control and Prevention (3).

To assess the influence of potential confounding variables on the results, we reestimated regression models with the following covariates added as explanatory variables: sex, ethnicity, socioeconomic status, whether the child had lived in a community with fluoridated water, use of toothpaste, age at which child began using toothpaste, brushing frequency, use of mouth rinse, use of fluoride

supplement, use of floss, time since last dental examination, dental hygiene rating, carbohydrate consumption, number of snacks per day, whether the child had been breast fed, and use of medications that cause dry mouth.

Caries on particular subsets of tooth surfaces were also analyzed, using the same methods. The subsets were chosen based on hypothetical mechanisms by which lead exposure may cause caries:

- A decrease in salivary flow impairs the buffering function of saliva in protecting the tooth against bacterial acids and leads to an increase in the prevalence of caries (22). A rat study reported that lead exposure is associated with a concurrent decrement in salivary flow (23). Because lead ingestion typically occurs at 1–3 years of age (13), we reasoned that this mechanism would more likely affect deciduous teeth rather than permanent teeth. Similarly, since lingual surfaces (surface of tooth facing the tongue) are in more contact with saliva than buccal surfaces (surface of tooth facing the buccal and labial mucosa) (24,25), this hypothesis also predicts that the lingual surfaces would be less affected by reduced salivary flow. Therefore, in addition to dfs and DMFS, we analyzed caries on deciduous lingual surfaces and deciduous buccal surfaces.
- It has been proposed that lead may bind with salivary fluoride and thus diminish its protective effect on enamel demineralization (26). Because fluoride is most effective in preventing caries on smooth surfaces (the sides of the teeth), and least on occlusal surfaces (the chewing surfaces) (27), we analyzed caries on smooth surfaces and occlusal surfaces separately. Again, because lead ingestion typically occurs at 1–3 years of age (13), we expected that this mechanism would affect deciduous teeth rather than permanent teeth and therefore conducted the analysis on deciduous teeth.

**Table 1.** Makeup of questionnaire.

Demographics
Head of household's occupation
Head of household's highest grade attained in school
Fluoride exposure
Name of communities the child has ever lived in
Age began, frequency, and type of toothpaste use
Age began, frequency, and type of mouthwash use
Age began, frequency, and type of fluoride supplement use
Diet
Whether consumed each of seven specified carbohydrates the previous day
Number of snacks per day
Oral hygiene
Time since last dental visit
Frequency of tooth brushing
Whether child flosses
Medical history
Presence of conditions that predispose to caries
Current medications

• Another proposed mechanism is that lead, incorporated into enamel during calcification, may render the enamel more susceptible to caries (28). Permanent teeth calcify between birth and 16 years of age (29). This age period includes the time during which blood lead levels tend to peak in children (13). We thus reasoned that permanent teeth would be more affected than deciduous teeth. However, several of the permanent teeth calcify outside the age range when children tend to ingest lead. Because the first permanent molars calcify between birth and age 36 months (29), an age period much more likely to overlap the age during which children tend to ingest lead, we analyzed caries on the first permanent molars.

## Results

A total of 1,660 children were examined for caries at their elementary schools during the 1995–1996 and 1996–1997 academic years.

**Table 2.** Comparison of variables between the primary sample and interviewed sample.

Variable	Primary sample	Interviewed sample
Sample size	248	154
Demographics		
Sex (% male)	50%	49%
Ethnic distribution		
African American	68%	69%
White	10%	10%
Hispanic	20%	18%
Asian	2%	3%
Lead measures		
Mean lead exposure ( $\mu\text{g}/\text{dL}$ ) <sup>a</sup>	10.7	11.5
Lead exposure range	0–45	2–45
Mean age at blood collection (months) <sup>b</sup>	27.3	27.5
Lead level range	18.0–36.8	18.0–36.7
Mean age at dental examination (years)	8.4	8.4
Age range	6.9–12.0	6.9–12.0
Grade at examination		
Second	79%	79%
Fifth	21%	21%
Hygiene score		
Good	3%	3%
Fair	90%	88%
Poor	7%	9%
Mean dfs <sup>c</sup>	3.4 <sup>d</sup>	4.2 <sup>e</sup>
dfs range	0–29	0–29
Mean deciduous SAR <sup>f</sup>	54.4 <sup>d</sup>	53.8 <sup>e</sup>
Deciduous SAR range	4–88	4–80
Mean DMFS <sup>g</sup>	0.5 <sup>h</sup>	0.5
DMFS range	0–8	0–8
Mean permanent SAR <sup>i</sup>	52.9 <sup>h</sup>	53.6
Permanent SAR range	8–128	8–128

<sup>a</sup>To convert to  $\mu\text{mol}/\text{L}$ , multiply by 0.0483. <sup>b</sup>Mean age at blood collection for blood lead level. <sup>c</sup>The number of decayed or filled surfaces in deciduous teeth. <sup>d</sup>Twenty children had no deciduous teeth and were excluded from the computation. <sup>e</sup>Fourteen children had no deciduous teeth and were excluded from the computation. <sup>f</sup>Deciduous surfaces at risk (equal to the total number of deciduous surfaces minus number of sealed deciduous surfaces). <sup>g</sup>The number of decayed, missing, or filled surfaces in permanent teeth. <sup>h</sup>One child had no permanent teeth and was excluded from the computation. <sup>i</sup>Permanent surfaces at risk (equal to the total number of permanent surfaces minus number of sealed permanent surfaces).

Of these children, 248 had 1 or more appropriate blood lead measurements. These children composed the primary sample for analysis. Table 2 lists the demographics, lead measures, and dental measures of this sample. Parents of 154 children (62%) completed a telephone interview, 8 (3%) parents declined the interview, and 86 (35%) could not be contacted. The demographics, lead measures, and dental measures of the 154 children whose parents completed the telephone interview are presented in Table 2; these measures were comparable to those for the entire primary sample of children.

Among the primary sample of children ( $n = 248$ ), the mean lead exposure was 0.52  $\mu\text{mol}/\text{L}$  (10.7  $\mu\text{g}/\text{dL}$ ) and ranged from 0 to 2.17  $\mu\text{mol}/\text{L}$  (0–45  $\mu\text{g}/\text{dL}$ ; Table 2). One hundred sixty-four (66%) children had a lead exposure < 0.48  $\mu\text{mol}/\text{L}$  (< 10  $\mu\text{g}/\text{dL}$ ), and 84 (34%) had a lead exposure  $\geq$  0.48  $\mu\text{mol}/\text{L}$  ( $\geq$  10  $\mu\text{g}/\text{dL}$ ). Twenty children had no deciduous teeth, 114 (50%) had a dfs = 0, and 114 (50%) had a dfs  $\geq$  1; the mean dfs was 3.4 and ranged from 0 to 29 (Table 2). One child had no permanent teeth, 199 (81%) had a DMFS = 0, and 48 (19%) had a DMFS  $\geq$  1; the mean DMFS was 0.5 and ranged from 0 to 8 (Table 2). These proportions of caries are comparable to national prevalence studies (5,6).

The proportions of children with caries in permanent teeth (DMFS  $\geq$  1) as a function of lead exposure [ $<$  0.48  $\mu\text{mol}/\text{L}$  vs.  $\geq$  0.48  $\mu\text{mol}/\text{L}$  (< 10  $\mu\text{g}/\text{dL}$  vs.  $\geq$  10  $\mu\text{g}/\text{dL}$ )] were 15% and 27%, respectively. The proportions of children with caries in deciduous teeth (dfs  $\geq$  1) as a function of lead exposure were 46% and 59%, respectively. However, children with lower lead exposure [ $<$  0.48  $\mu\text{mol}/\text{L}$  (< 10  $\mu\text{g}/\text{dL}$ )] were younger on average than those with higher lead exposure (8.1 years vs. 9.1 years, respectively;  $p < 0.001$ ), more likely to be in second grade (90% vs. 60%, respectively;  $p < 0.001$ ) and had fewer permanent surfaces at risk (means 47.0 vs. 64.3, respectively;  $p < 0.001$ ). Therefore, we included age

**Table 3.** Adjusted odds ratios for association of caries with lead exposure [ $<$  0.48  $\mu\text{mol}/\text{L}$  vs.  $\geq$  0.48  $\mu\text{mol}/\text{L}$  (< 10  $\mu\text{g}/\text{dL}$  vs.  $\geq$  10  $\mu\text{g}/\text{dL}$ )]<sup>a</sup>.

Caries measure	OR	95% CI
Permanent teeth		
DMFS <sup>b</sup>	0.95	0.43–2.09
First molar	1.12	0.52–2.39
Deciduous teeth		
dfs <sup>c</sup>	1.77	0.97–3.24*
Occlusal surfaces	1.43	0.78–2.60
Smooth surfaces	1.44	0.80–2.62
Lingual surfaces	2.31	1.18–4.51**
Buccal surfaces	1.51	0.72–3.15

<sup>a</sup>Adjusted for age at examination, grade in school, and the number of surfaces at risk. <sup>b</sup>The number of decayed, missing, or filled surfaces in permanent teeth. <sup>c</sup>The number of decayed or filled surfaces in deciduous teeth. \* $p = 0.07$ . \*\* $p = 0.01$ .

at exam, grade in school, and surfaces at risk as covariates in all the logistic regression analysis relating caries to lead exposure.

Table 3 reports the adjusted odds ratios for the probability of caries in children with high versus low lead exposure. For the DMFS measure, the odds ratio was 0.95 [95% confidence interval (CI), 0.43–2.09;  $p = 0.89$ ]. For the dfs caries measure, the odds ratio was 1.77 (95% CI, 0.97–3.24;  $p = 0.07$ ). The only statistically significant association was for caries on the deciduous lingual surfaces, for which the odds ratio was 2.31 (95% CI, 1.18–4.51;  $p = 0.01$ ). Adding the patient characteristics listed in the previous section to the regressions did not strengthen the estimated associations between lead exposure and caries.

## Discussion

This study identified some paths for future study, and other avenues that do not look promising. We found that the odds ratio relating caries on deciduous teeth to lead exposure was marginally significant ( $0.05 < p < 0.10$ ); the odds ratio for caries on deciduous lingual surfaces was significant at the 0.05 level (Table 3). None of the remaining comparisons were significant. Despite these significant findings, we did not see patterns of caries consistent with postulated mechanisms. For example, the hypothesis that lead, incorporated into enamel during calcification, renders the enamel more susceptible to caries would suggest a stronger association between lead exposure and caries in permanent teeth (DMFS) than in deciduous teeth (dfs). However, this pattern was not seen. Also, the hypothesis that lead causes a decrease in salivary flow would suggest a stronger association between lead exposure and caries in deciduous teeth than permanent teeth. Although this pattern was seen, the deciduous lingual surfaces were more affected than deciduous buccal surfaces—the opposite of what was expected. Therefore, the most conservative interpretation of the data is that this study failed to demonstrate a strong association between lead exposure and caries.

However, there are three limitations that may have caused an underestimation of any association. First, the lead exposure measure, in many cases based on a single blood lead level, was not a precise measure of cumulative lead exposure. Blood lead levels drawn after age 37 months were not included because studies have shown that most children are exposed to lead before this age (13). However, had lead exposure increased after age 37 months, we would be underestimating the true lead exposure, and such a misclassification bias would underestimate the odds ratios. Second, because the subjects were examined at a young age (the mean age at examination was 8.4 years), the caries

experience of the permanent teeth was low (the mean DMFS score was only 0.5). Therefore, sufficient time may not have elapsed for caries to become manifested on permanent teeth. Moss et al. (30), in a secondary data analysis of the NHANES III, reported a significant association between lead exposure and caries on permanent teeth of an older cohort of children aged 5–17 years. Finally, the purpose of the school-based caries-screening program was to identify cavitory lesions that required treatment, not to identify noncavitory, precarious lesions. This will underestimate the prevalence of caries (31).

Another limitation of this study was that it lacked statistical power to detect a low yet clinically relevant odds ratio. The secondary data analysis by Moss et al. (30) reports odds ratios of 1.36–1.66 for caries in permanent teeth. Although they appear to be low, such odds ratios imply a sizable burden of disease: an additional 2.7 million children may have caries as a result of lead exposure (30). The odds ratios reported by Moss et al. are consistent with our data, in that they are included in our confidence intervals. However, with a sample size of 248 subjects, we had less than 30% power for detecting an odds ratio of  $\leq 1.5$ .

In summary, this study did not demonstrate an association between lead exposure and caries. However, due to the limitations described above, the findings do not exclude the possibility that such an association exists.

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